residues in lungs from nonoccupationally exposed persons would be well worthwhile. The recently reported data of Whitwell and associates point up the great potential value of such studies.

In conclusion, there is no question of the need for appropriate hygiene controls when asbestos products are being possessed or manipulated in ships afloat or in the industrial setting. These are, in fact, occupational exposures, and all of the necessary precautions associated with such activity are warranted and should be mandatory. In this manner, exposure of those not involved directly should be greatly lessened or abolished. On the other hand, I believe there is evidence to support the contention that there is little or no overriding need for immediate systematic removal of asbestos *in situ*, except where it can be demonstrated to create uncontrollable exposure approximating the levels now regulated occupationally. Direct systematic study of the exposures and of the health experiences of those who have served and continue to serve aboard ship or in industry is needed.

ASBESTOSIS AMONG HOUSEHOLD CONTACTS OF ASBESTOS FACTORY WORKERS*

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Equally ubiquitous in the environment as industrial chemical wastes[1] are inorganic microparticles such as asbestos. The environmental burden of asbestos pollution is a recent phenomenon which has grown with the rapid expansion of asbestos-utilizing industries.[2] The health consequences of poorly controlled *occupational* exposures to chemicals and dusts now found in the general environment have been known in many instances for well over 100 years.[3] Reports of overt disease (usually seen only with occupational exposure) among nonoccupationally exposed individuals have frequently been considered medical curiosities when they appeared in the medical literature. However, the full extent of the health risks due to nonoccupational exposure to toxic agents is not known, for it is uncommon to inquire into the neighborhood residence history or occupation and exposures of a patient's household contacts when investigating symptoms of a disease. The effects of such exposures may be mild or subclinical manifestations which are only contributory to a current health problem and their role goes unrecognized.

In 1976, we reported on a systematic investigation of one such non-occupational exposure to asbestos dust.[4] The group studied consisted of household contacts of workers in an asbestos factory manufacturing amosite asbestos insulation materials between 1941 and 1954. None of those reported had personal occupational exposure to asbestos. Yet 35% had asbestos-associated radiographic abnormalities. A source of home contamination in individual exposure was postulated as resulting from dust adhering to shoes, hair, and workclothes brought home for laundering. Changerooms and company laundered coveralls were not available at this plant. We reported the identification of four pleural mesotheliomas among the family contacts of the 1,664 workers who were employed at some time by the factory. Since that time one additional pleural mesotherlioma death has occurred, raising the total mesothelioma deaths to date among the group under observation to five.

This report extends the continuing clinical investigation of this cohort.

Population Studied

We are actively investigating the status of household contacts of the 1,664 asbestos workers employed in the factory which produced amosite asbestos products from 1941-1954. The names of household contacts are obtained from the surviving worker cohort members and from other sources for those already deceased. Once identified as

*This research is supported by National Institute for Environmental Health Sciences Grant No. ES 00929 and a grant from the National Cancer Institute, CA 22792.

a possible household contact, a variety of epidemiologic tracing techniques is utilized to locate the individual and ascertain his/her current status. Living individuals are invited to participate in periodic medical clinics.

Control Population

For comparison purposes in the chest x-ray interpretation, a control population was felt to be appropriate. Urban New Jersey residents currently living in the same community as the study population were identified from the general medical clinic at the facility used to examine the workers and household members. Consecutive eligible individuals who appeared for routine chest x-rays between January 1, 1975 and December 31, 1976 were selected until age and sex classes similar to the first 326 examined household contacts were filled; thus, the age and sex distribution of the study group and controls were similar. Clinical records of all controls were reviewed and only those with no recorded personal occupational contact with asbestos were included. Only PA films were available for review. No new clinical examinations or spirometry were performed on controls.

Methods

The clinical examination includes comprehensive lifetime occupational and residential histories, past and current medical history, history of current symptoms, smoking and respiratory questionnaires, physical examination, 14″ X 17″ posteroanterior, right and left oblique chest x-rays, and spirometry.

After each clinic, the chest x-rays of household contacts, factory workers and previously identified controls were interspersed and read without knowledge of exposure category. The ILO U/C Pneumoconioses Classification of 1971 was utilized by a panel of five experienced readers.[5] A consensus interpretation was arrived at and it is this reading that was utilized for statistical analyses. A film was classified as abnormal if one or more of the following were recorded: small opacities (combined rounded and irregular) 1/0 or more; any lateral wall pleural thickening; any pleural calcification; pleural plaques on the diaphragm. Although recorded, blunting of the costophrenic angles was not included in this analysis. Only abnormalities visible on the PA film were utilized. The added utility of the oblique films will be discussed in a later report.

The actual extent and intensity of each household resident's asbestos exposure is unavailable. To help approximate a relative exposure dose index for each household contact, the employment history of the factory worker in whose household he/she resided was utilized. The resident *worker* for each household is referred to as the "index worker." Thus, each household contact was assigned a "duration of exposure" which was equivalent to the length of time lived in the household while a worker was actively employed. Similarly, each contact was assigned a year of "onset of exposure" equivalent to the year in which he/she first resided with an actively employed worker.

Statistical Analyses

Prevalence data were compared by the χ^2 test. Differences between means were compared using Student's t-test. P values at less than .05 were considered significant. Statistical analyses were performed with the cooperation of the University of New York Computer Center, using the Statistical Package for the Social Sciences.[6]

Results

In the six-year period 1973–1978, over 3100 household contacts of the 1664 amosite asbestos factory workers were identified. Of that group, 756 are known to be deceased, and of the remaining 2300+, 771 have been examined in our clinics. During 1975–1976, 326 controls were identified.

Ninety-two individuals did not meet the criteria set for inclusion in this analysis. Five household contacts and one control are not included in this report because of current medical conditions which are known to cause the types of x-ray abnormalities seen on their radiographs. The use of the ILO U/C Classification would thus be misleading. These included three pleural mesotheliomas, one lymphosarcoma (post radiation and surgical therapy), two cases of sarcoidosis (one in a control). In addition, there were two unreadable, under-exposed films and one examinee refused a chest x-ray. Residence histories were carefully reviewed and individuals who were not actually in residence with the index worker at the time he was actively employed, were placed in a separate category. Thirty-three individuals were placed into this subgroup. They resided in the household only *after* the worker had ceased active employment. Occupational histories taken at the time of examination revealed that 51 individuals had potential personal past occupational exposure to asbestos. The distribution of occupations of these individuals and the prevalence of radiographic abnormality is shown in Table 1. Thus, 92 of the 771 examined did not meet the criteria for inclusion in the study, which is limited to the 679 household contacts who had lived in the household of an actively employed amosite asbestos factory worker and who themselves had not had an occupational exposure to asbestos or other fibrogenic dust.

Table 1

Occupational Exposures to Asbestos among 756 Household Contacts

Occupation	Number	Abnormal X-ray
Direct asbestos trade	19	7 (37%)
Brake repair work	8	4 (50%)
Dry wall construction	1	0
Shipyard work	6	4 (67%)
Indirect asbestos exposure	16	8 (50%)
Other fibrogenic dust exposure	1	0
Total	51	23 (45%)

The age distribution for the study subjects and the control population showed that the controls were slightly older than the household contacts (mean age 48 compared to 45 in the contacts) (Table 2).

The distribution of examined household contacts by the duration of their exposure in the home of an actively employed index worker and by the year that they were first exposed is shown in Table 3. Duration of active employment and presumably concomitant active daily household contamination was less than one year for 28% of those examined. In only 5% was index worker employment, with increasing household contamination, 10 or more years. At the time of their examination, all study participants were more than 20 years from onset of their first asbestos exposure.

TABLE 2

AGE DISTRIBUTION OF HOUSEHOLD CONTACTS AND CONTROLS AT TIME OF X-RAY EXAMINATION

Age (yrs.)	Number of Household Contacts*	Number of Controls†
20–29	54 (8%)	30 (9%)
30–39	188 (28%)	59 (18%)
40–49	196 (29%)	80 (25%)
50–59	144 (21%)	83 (26%)
60–69	68 (10%)	51 (16%)
70+	29 (4%)	22 (7%)
Total	679	325

*Mean age of household contacts 45.7 ± 12 yrs.
†Mean age of controls 48.6 ± 14 yrs.

Altogether nearly one-third of those examined were under the age of 40. Two-thirds of individuals had already exceeded 30 years from onset of initial asbestos exposure. Information is not available on the dimensions of the asbestos contamination which remained in the house after the worker changed employment.

The prevalence of radiographic abnormalities among the survey participant groups is summarized in TABLE 4. The control population had a 5% overall prevalence of radiographic abnormalities. Individuals who resided in a household *after* the index worker's employment period had the second lowest prevalence of abnormalities (12%) followed by persons in residence (35%) *during* the index worker's employment. As would be expected, the highest prevalence of abnormalities (45%) was among the household residents with reported personal occupational asbestos exposure. Pleural abnormalities were more prevalent than parenchymal small opacities.

When the comparative prevalence of the various types of asbestos-associated radiographic abnormalities was analyzed, it was found that pleural abnormalities were the most prominent (TABLE 5). Pleural calcifications were present in 8% of the household contacts and were seen in none of the controls. Pleural thickening was present on 19% of the household contact radiographs and only 1% of the control chest x-rays. Overall, 239 (35%) of the household contacts had one or more abnormalities present compared to 15 (5%) of the control group. These radiographic abnormalities

TABLE 3

DISTRIBUTION OF HOUSEHOLD CONTACTS BY DURATION OF EXPOSURE AND YEAR OF ONSET OF 1ST EXPOSURE

Year of First Exposure	Total Examined	Duration of Exposure			
		<1 Year	1–5 Years	5–10 Years	10+ Years
1941–46	436	124	246	32	34
1946–50	113	29	68	16	—
1950–55	129	40	89	—	—
Total	678	193 (28%)	403 (59%)	[illegible]%)	34 (5%)

TABLE 4

PREVALENCE OF ASBESTOS ASSOCIATED RADIOGRAPHIC ABNORMALITIES AMONG SURVEY PARTICIPANT GROUPS*

Exposure Group	Total Examined	Small Opacities	Pleural Abnormality		Small Opacities and/or Pleural Abnormalities	
Urban New Jersey residents (controls)	325	10 (3%)	6 (2%)	$\chi^2 = 6.4$ $p < .02$	15 (5%)	$\chi^2 = 114.$ $p < .001$
Household resident *after* index worker employment period	33	1 (3%)	3 (9%)		4 (12%)	
Household resident during index worker employment period	679	114 (17%)	178 (26%)	$\chi^2 = 7.5$ $p < .01$	239 (35%)	
Household resident and personal occupational asbestos exposure	51	13 (25%)	18 (35%)		23 (45%)	

*ILO U/C Pneumoconiosis Classification small opacities (combined) 1/0 or greater; pleural thickening; pleural calcification; pleural plaques.

were seen in various combinations (TABLE 6). Pleural change as the only abnormality on the chest x-ray was seen in 18% of the household contacts. This is compared to 9% with no pleural changes and only parenchymal small opacities present. In 8% of household contacts, both small opacities and a pleural abnormality were present. Among the controls, small opacities as the only abnormality present was the most prevalent (3%). Only one of the controls (0.3%) had both small opacities and a pleural abnormality present.

The distribution by profusion of combined small opacities (graded according to the ILO U/C Classification) among the household contacts and controls is shown in TABLE 7. Only two of the household contacts (0.3%) had a category 2 profusion of combined small opacities. Forty-seven of the 114 films with small opacities were in the lowest category, 1/0. Eight out of the ten control films were coded as having combined small opacities in the lowest category.

Pleural thickening and pleural calcification were seen in all extent categories (TABLES 8 and 9). As with the parenchymal abnormalities, the pleural abnormalities in household contacts tended to be classified in the lower ILO U/C categories. However, 19 (3%) of household contacts had extensive pleural thickening and 10 (2%) had extensive pleural calcification present.

TABLE 5

PREVALENCE OF ASBESTOS-ASSOCIATED RADIOGRAPHIC ABNORMALITIES AMONG HOUSEHOLD CONTACTS OF AMOSITE ASBESTOS WORKERS AND CONTROLS

Group	Total Examined	Small Irregular Opacities	Pleural Thickening	Pleural Calcification	Pleural Plaques	One or more Abnormalities
Household contacts	678	114 (17%)*	128 (19%)*	54 (8%)*	57 (8%)*	239 (35%)*
Controls	325	10 (3%)	4 (1%)	0	2 (0.6%)	15 (5%)

*Prevalence of all types of abnormalities significantly higher in the household contacts than the controls, $p < .001$.

The possible effect of increasing duration of exposure as an index of increasing dose was also examined. Among occupational groups, duration of exposure correlates well with increasing prevalence of radiographic abnormalities. The prevalence of radiographic abnormalities among the household contacts by duration of exposure is presented in TABLE 10. All abnormalities show an increase in prevalence as duration of exposure increases. The category of combined small opacities did not show a statistically significant increase in prevalence with duration of exposure although a trend is apparent. For pleural thickening, not only did the prevalence of the abnormality increase with duration of exposure but also the extent of disease (TABLE 8). However, this did not seem to be the case for pleural calcification (TABLE 9).

Under occupational exposure conditions, for a majority of workers, a period of clinical latency of approximately 20 years from onset of exposure to appearance of radiographically detectable asbestos-associated disease has been well documented.[7] All household contacts had exceeded the 20 year from onset point at the time of their initial examination. Individuals first exposed between 1941 and 1946 had the highest prevalence of abnormalities (TABLE 11). The group exposed between 1950 and 1954 had the lowest prevalence of abnormalities (22%). The increasing prevalence of radiographic abnormality with longer time since onset of first exposure is most evident

TABLE 6

PREVALANCE OF RADIOGRAPHIC ABNORMALITY (COMBINATIONS) AMONG HOUSEHOLD CONTACTS AND CONTROLS*

Group	Number Examined	Small Opacities Only	Pleural Thickening Only	Pleural Calcification Only	Pleural Plaques Only	More than One Pleural Abnormality	Small Opacities and Pleural Abnormality	Total Abnormal (Pleural and/or Parenchyma)
Household contacts	679	61 (9%) a	70 (10%) a	16 (2%) a	18 (3%) b	21 (3%) a	53 (8%) a	239 (35%) a
Controls	325	9 (3%)	3 (1%)	0	2 (0.6%)	0	1 (0.3%)	15 (5%)

*Prevalance of radiographic abnormalities significantly greater among household contacts than controls. a – $p < .01$; b – $p < .05$.

TABLE 7

DISTRIBUTION OF PROFUSION OF COMBINED SMALL OPACITIES AMONG HOUSEHOLD CONTACTS AND CONTROLS

Group	Total Examined	Combined Small Opacities					Combined Opacities
		0/0	0/1	1/0	1/1	1/2	2/2	1/0 or Greater
Household contacts	677	398 (59%)	165 (24%)	47 (7%)	61 (9%)	4 (1%)	2 (0.3%)	114 (17%)
Controls	325	288 (89%)	27 (8%)	8 (3%)	2 (0.6%)	0	0	10 (3%)

TABLE 8

PREVALENCE OF PLEURAL THICKENING BY DURATION OF HOUSEHOLD CONTACT

Duration of Exposure (yrs.)	Total Examined	Extent of Pleural Thickening (ILO U/C Classification) Extent 1	Extent 2	Total Pleural* Thickening
<1	192	15 (8%)	1 (0.5%)	16 (8%)
1-5	403	70 (17%)	7 (2%)	77 (19%)
5-10	48	17 (35%)	5 (10%)	22 (46%)
10+	34	7 (21%)	6 (18%)	13 (38%)
Total	677	109 (16%)	19 (3%)	128 (19%)

*Prevalence of pleural thickening significantly higher among those with longer duration of exposure.
$\chi^2 = 45$ $p < .001$ $df = 3$.

TABLE 9

DISTRIBUTION OF PLEURAL CALCIFICATION BY EXTENT* AND DURATION OF EXPOSURE

Duration of Exposure (yrs.)	Total Examined	Extent of Pleural Calcification Grade 1	Grade 2	Grade 3	All† Grades
<1	192	5 (3%)	1 (0.5%)	1 (0.5%)	7 (4%)
1-5	403	12 (3%)	14 (4%)	8 (2%)	34 (8%)
5-10	48	6 (13%)	2 (4%)	1 (2%)	9 (19%)
10+	34	4 (12%)	0	0	4 (12%)
Total	677*	27 (4%)	17 (3%)	10 (2%)	54 (8%)

*Two individuals with uncertain duration of exposure excluded.
†Prevalence of pleural calcification significantly greater among those with longer duration of exposure.
$\chi^2 = 12$ $p < .01$ $df = 3$.

TABLE 10

PREVALENCE OF RADIOGRAPHIC ABNORMALITIES AMONG HOUSEHOLD CONTACTS BY DURATION OF EXPOSURE*

Duration of Exposure (yrs.)	Number Examined	Small Opacities	Pleural Thickening	Pleural Calcification	One or More Abnormality
<1	192	27 (14%)	16 (8%)	7 (4%)	47 (24%)
1-5	403	65 (16%)	77 (19%)	34 (8%)	145 (36%)
5-10	48	14 (29%)	22 (46%)	9 (19%)	29 (60%)
10+	34	8 (24%)	13 (38%)	4 (12%)	18 (53%)
Total	677	114 (17%)	128 (19%)	54 (8%)	239 (35%)

*Prevalence of pleural thickening, pleural calcification and all abnormalities significantly higher among household contacts with longer duration of exposure (p).

TABLE 11

PREVALENCE OF RADIOGRAPHIC ABNORMALITIES BY YEAR OF ONSET OF EXPOSURE*

Year of Onset of Exposure	Total Examined	Small Opacities	Pleural† Thickening	Pleural† Calcification	One or more† Abnormalities
1941-1946	434	79 (18%)	98 (23%)	46 (11%)	172 (40%)
1946-1950	113	19 (17%)	21 (19%)	5 (4%)	38 (34%)
1950-1955	129	16 (12%)	9 (7%)	3 (2%)	29 (22%)
Total	676*	114 (17%)	128 (19%)	54 (8%)	239 (35%)

*Three individuals with uncertain year of onset excluded.
†Prevalence of abnormality greater among those with earlier onset of exposure. ($p < .01$).

for the pleural abnormalities. Statistical significance was not reached for a similar increase in combined small opacities.

The relationship of the household contact to the worker was also important (TABLE 12). Wives had the highest prevalence of abnormalities (48%) and daughters had the lowest (21%). Mean duration of exposure was the same for all relationship categories. The prevalence of abnormalities was statistically significantly lower among the daughters when compared to the other exposure groups.

With only a few exceptions, household contact clinic participants were unaware that they had any asbestos-associated disease. The prevalence of other disease processes was unrelated to asbestos exposure (arthritis, diabetes, hypertension, gout, etc.). The household contacts considered themselves to be in good health; the clinical examinations generally confirmed this.

Current symptoms and clinical findings detected among the household contacts examined are summarized in TABLE 13. Rhonchi were the most common physical findings, not significantly associated with the prevalence of any type of radiographic abnormality. On the other hand, dyspnea, rales, cyanosis and clubbing were significantly more prevalent among those with small opacities and/or pleural abnormalities on their x-rays.

TABLE 12

PREVALENCE OF RADIOGRAPHIC ABNORMALITIES BY HOUSEHOLD CONTACT'S RELATIONSHIP TO INDEX WORKER

Relationship	Total Examined	Small Opacities	Pleural Abnormality	Parenchymal and/or Pleural	Mean Duration of Exposure
Wives	162	40 (25%)	58 (36%)	77 (48%)	2.2 ± 2.4 yrs.
Daughter	224	15 (7%)	34 (15%)	46 (21%)*	2.4 ± 2.6 yrs.
Sons	151	31 (21%)	47 (31%)	63 (42%)	2.3 ± 2.8 yrs.
Siblings	81	15 (19%)	22 (27%)	30 (37%)	2.1 ± 2.1 yrs.
Others	61	12 (21%)	17 (28%)	23 (38%)	3.6 ± 3.3 yrs.
Total	679	129 (19%)	178 (26%)	239 (35%)	

*Prevalence of radiographic abnormalities significantly higher among sons than daughters.
$\chi^2 = 19$) .001.

Discussion

We have examined nearly one-third of the living household asbestos contact cohort members. New data confirm and extend our previous observations. The presence of radiographic abnormalities in our study cohort was statistically significantly associated with clinical abnormalities observed, duration of index worker employment and length of time since first exposure. These x-ray abnormalities, which are usually seen only after occupational asbestos exposure, were significantly more prevalent among the study group than a control comparison group. Although the number of examined individuals has more than doubled since our previous report, the overall prevalence of abnormalities has remained constant. It is most probable that our results represent a good approximation of the prevalance of radiographic abnormalities in the total cohort.

Table 13

Prevalence of Current Symptoms and Clinical Findings* among Household Contacts by Radiographic Abnormalities†

Radiographic Finding	Number Examined	Dyspnea	Rales	Rhonchi	Cyanosis	Clubbing
Small opacities	Absent 558	22 (4%)	23 (4%)	27 (5%)	2 (0.4%)	2 (0.4%)
		§	‡	¶	¶	§
	Present 111	11 (10%)	11 (10%)	10 (9%)	2 (2%)	3 (3%)
Pleural change	Absent 495	20 (4%)	18 (4%)	26 (5%)	0	2 (0.4%)
		¶	§	¶	§	¶
	Present 174	13 (8%)	16 (9%)	11 (6%)	4 (2%)	3 (2%)
Small opacities and/or pleural abnormality	Absent 435	14 (3%)	13 (3%)	21 (5%)	0	1 (0.2%)
		§	§	¶	§	
	Present 234	19 (8%)	21 (9%)	16 (7%)	4 (2%)	4 (2%)
Total	669	33 (5%)	34 (5%)	37 (6%)	4 (0.6%)	5 (0.7%)

*Ten individuals did not have physical examinations.

†Prevalence of abnormality significantly greater among those with radiographic abnormality than those with normal x-rays.

‡Significantly different at $p < .05$ using χ^2 test.

§Significantly different at $p < .01$ using χ^2 test.

¶Not statistically significant.

The early observation that pleural calcification, which is uncommon in the general population (.02–.5%)[8-10] and common in asbestos-exposed populations (30+%)[7,11] suggested that pleural calcification might serve as a marker of past asbestos exposure in non-occupationally exposed groups. This association was first remarked upon by Kiviluoto in 1960 when he described an abnormally high prevalence of pleural calcification among residents of a district in Finland in which an anthophyllite asbestos mine and mill had been operating for many years.[12] Subsequently, similar observations were made in other countries.[13-15] A survey of factory workers, factory worker household contacts, and factory neighborhood residents reported in 1971 by Navratil described an increased prevalence of pleural calcification among not only the factory workers but the relatives of factory workers and the residents in the neighborhood of the factory.[16] However, both the neighborhood and bloc[illegible]ative groups were small (155 and 114 respectively).

Our continuing survey extends the observations of these authors and indicates that pleuro-pulmonary disease can occur under the conditions of nonoccupational exposure as experienced in the homes of asbestos workers. Of the household contacts with occupational exposure to asbestos, only 19 were aware that they were or had been working with asbestos. Individuals who reported having done brake repair work in the past, worked in shipyards, or performed general maintenance work involving pipe insulation, had the highest prevalence of abnormal x-rays (more than 50%). Of special interest is the group of household residents who entered the index worker's home shortly *after* his active employment period had ended. This group of 33 individuals did not show an increased prevalence of small opacities. However, they did have a statistically significantly increased prevalence of pleural abnormalities when compared to the urban New Jersey resident controls. It would appear that pleural changes are a better indicator of environmental asbestos exposure than are parenchymal small opacities.

The proportion of household contacts who had the more classic appearance of asbestosis, with both parenchymal small opacities and pleural abnormalities, was small (Table 6). This again indicates the importance of assessing pleural disease in nonoccupational settings.

The appearance of asbestos-associated disease has been shown to depend upon duration and intensity of exposure. The longer and more intense the exposure, the sooner the disease could be expected to appear and, conversely, the shorter and less intense the exposure, the longer the time (latency) before an increased prevalence of disease could be demonstrated. As in occupational groups, increasing duration of exposure was associated with a higher prevalence of abnormalities in our study group. The effects of duration of exposure and time since onset of exposure were most marked for the pleural abnormalities. Individuals with more than ten years of exposure had four times as much pleural thickening and three times as much pleural calcification as individuals with less than one year of exposure. There was less than a two-fold increase in the prevalence of combined small opacities between the two groups. Forty percent of individuals first exposed between 1941 and 1946 had an abnormal x-ray compared to only 22% for those exposed first between 1950 and 1954. The data presented here demonstrate the time dynamics for the appearance of radiographic abnormalities after household asbestos exposure. It appears that there is a longer period of latency between first exposure and appearance of radiologic abnormalities under the conditions of household asbestos exposure.

The observation that sons have twice the prevalence of radiographic abnormalities of daughters is an interesting one. One possible explanation for the difference would be that the sons had a longer duration of exposure. However, it can be seen from Table 12 that the sons and daughters had the same mean duration of exposure. Further investigation of these differences is being made. The observation that nearly one-half of the wives examined had abnormal x-rays is consistent with the hypothesis that the wives would have been most heavily exposed because they were responsible for the laundering of workclothes and resided in the household for the longest period of time.

Dyspnea, which is often the initial complaint of occupationally exposed asbestos workers, was present in 10% of individuals with parenchymal small opacities on their chest x-rays compared to only 4% of individuals with normal chest x-rays. No statistical association between dyspnea and pleural changes was seen although a similar trend was apparent. Dry rales, commonly heard in asbestotic patients, were also more prevalent among the household contacts with abnormal chest x-rays. Nine percent of [illegible] individuals with abnormal x-rays had rales present on physical examinatio[illegible]ompared to only 3% of individuals with normal x-rays. Rhonchi,

commonly associated with chronic bronchitis in cigarette smoking were not significantly associated with the presence or absence of radiographic abnormalities. Clubbing and cyanosis, associated with advanced disease, were uncommon and were found in only 5 and 4 individuals respectively.

Using only one measure of asbestos effect, the chest x-ray, we have seen in our cohort of household asbestos contacts that x-ray abnormalities characteristically seen only after occupational asbestos exposure, are common. We do not yet know the full extent of the disease risk which will be experienced by this cohort. We do know from our own studies and those of others, that household asbestos contact as well as other environmental exposure is associated with an increased risk of mesothelioma, especially pleural mesothelioma.[5,17,18] In our previous report, we described four instances of pleural mesothelioma from among the total household contact cohort under observation. An additional case has been detected. Although tracing is not complete, we have so far recorded slightly over 550 deaths and 5 of them are due to pleural mesothelioma.

Vianna and Polan recently reported a retrospective study of 52 cases of mesothelioma among women in New York State, 1967–73.[19] Using occupational histories gathered on the women's husbands and other household residents, they were able to calculate a relative risk factor of 10 for residents in the home of a worker in an asbestos related industry. Nine of the 52 cases reviewed had only household contact with asbestos.

Completion of the mortality survey of our cohort and continued prospective observation will help determine the extent of risk for malignant diseases which attends such household asbestos contact exposure.

The widespread use of asbestos and the subsequent release of fibers into the environment did not occur on a large scale until the mid-1940s. While the disease risk among the occupationally exposed workers during that period has already become apparent, we can expect that household contacts may not begin to manifest the possible effects of that exposure until 30 or more years from onset. In the next years we should begin to learn whether the levels of asbestos exposure experienced by groups such as the household contacts we have under observation will be sufficient to cause disabling disease other than mesothelioma.

It is essential that clinicians become aware of the significance of pleural disease unaccompanied by evidence of parenchymal fibrosis. In such cases, possible history of exposure to asbestos should be sought and the individual notified of the existence of the abnormality.

Acknowledgments

We are grateful for the valuable assistance given by our colleagues in the Environmental Sciences Laboratory in conducting the clinical studies and interpreting the chest x-rays, to our field survey staff, including Mrs. Dorothy Perron, Mr. Charles Nolan, Mrs. Rayla Margoles, Mrs. Stella Przybyla and Ms. Shirley Levine. We wish to thank Mrs. Selma Annenberg and Mr. Sidney Sibel for the preparation of tables and manuscript and Mr. James Fulmer for his assistance in the tabulation of the data.

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